

## Annotation

# Toddler's diarrhoea

A syndrome variously known as toddler's diarrhoea, chronic nonspecific diarrhoea, or the irritable colon syndrome of infancy is now recognised to be the most common cause of chronic diarrhoea without failure to thrive in early childhood.<sup>1-4</sup>

This syndrome generally has its onset between ages 6 and 24 months. It is a self-limiting disorder, usually ceasing spontaneously between ages 2 and 4 years, but occasionally it may persist beyond age 4.<sup>3</sup> Often the child presenting with diarrhoea has previously been constipated and sometimes has had infantile colic. The stool pattern is typically a large stool early in the day, formed or partly formed, followed by the passage of small loose stools containing undigested vegetable material and mucus, but this is not always the pattern and sometimes each stool is loose. This passage of recognisable undigested food in the stools is characteristic; indeed one popular name stemming from this observation is 'the peas and carrots syndrome'. A severe napkin rash may accompany this diarrhoea. Despite the presence of chronic diarrhoea, the child grows and develops normally. Although the child is otherwise healthy, many mothers become unduly preoccupied with each and every stool that the child passes. This may result in considerable maternal anxiety, family disharmony, and even marital discord.

### Aetiology

The nature of this common and self-limiting syndrome is not clear. There has been recent interest in it and a number of interesting observations have shed some light on its pathogenesis. The diarrhoea is not associated with malabsorption but is related to a decreased mouth-to-anus transit time in an otherwise healthy child.<sup>4</sup>

Although the small intestinal mucosa in these children is morphologically normal, Tripp *et al.*<sup>5</sup> found evidence of a significant increase in specific enzyme activity for adenylyl cyclase, and also for  $\text{Na}^+\text{K}^+$ -ATPase in small intestinal biopsies taken from children with this syndrome. They suggested that this increase was a response of normal villous cells to crypt cell secretion. This could be mediated via prostaglandins since high plasma

prostaglandin  $\text{F}\alpha$  levels have been found in some such children.<sup>6</sup> Cohen *et al.*<sup>3</sup> also reported that many of these children have had gastroenteritis or an acute illness immediately preceding or precipitating the onset of chronic diarrhoea. This observation accords with that of Tripp *et al.*<sup>5</sup> who found enzymic changes similar to those observed in toddler's diarrhoea in the small intestinal mucosa of children in the recovery phase from the postenteritis syndrome. Cohen *et al.*<sup>3</sup> found that some children presenting with this syndrome have a low intake of dietary fat. They claimed that an increased fat intake in such children might lead to a complete resolution of their symptoms. They suggested that the mechanism whereby fat consumption alters diarrhoea was mediated via an effect on motility.

It is important to differentiate toddler's diarrhoea from cows' milk-sensitive enteropathy, where the small mucosa is characteristically abnormal, and also from multiple food allergy. Small intestinal biopsy can be helpful but it may not discriminate toddler's diarrhoea from children with multiple food allergy; in this latter group serum IgE is typically raised, specific radioallergosorbent (RAST) tests are positive, and there is often eosinophilia.<sup>6</sup> These abnormalities are not a feature of children with toddler's diarrhoea.

Toddler's diarrhoea should only be considered as the diagnosis when the child is otherwise thriving and in good general health. In other children chronic diarrhoea may be part of a spectrum of familial functional bowel disorders with continuing gastrointestinal complaints which may present later.<sup>3</sup>

### Management

Treatment at the moment ranges from reassurance and explanation on the one hand to prescription of drugs on the other.

Hamdi and Dodge<sup>7</sup> showed that loperamide gave symptomatic benefit to some children with this syndrome. It was as effective in those with raised prostaglandin levels as those without. The widespread use of antidiarrhoeal drugs in paediatrics is to be deplored, but occasionally a child with a severe form of this syndrome benefits from a course of

loperamide. In fact it may be his mother who benefits the most! Such therapy should be given for a limited period only. Elimination diets of any kind are not indicated and their use in this syndrome is to be discouraged.<sup>8</sup> As fruit and vegetables are recognisable in stools these are sometimes excluded, but such a restriction is of no value. Indeed Cohen *et al.*<sup>3</sup> recommended increasing dietary fat intake to 4 kg of body weight/day when this was low. Since it is probable that such a low intake of fat is the result of previous professional dietary advice that the family has been given, it is fascinating to speculate that this syndrome may possibly be iatrogenic in some children.

Did these children have the postenteritis syndrome for which a milk-free, low fat diet had been prescribed, which in turn has perpetuated their chronic diarrhoea? It is obvious that more research is required to unravel the pathogenesis of this syndrome.

#### References

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- <sup>6</sup> Syme J. Investigation and treatment of multiple intestinal food allergy in childhood. In: Pepys J, Edwards A M, eds. *The mast cell*. Tunbridge Wells: Pitman Medical, 1979: 438-43.
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